

L7 ANSWER 134 OF 137 MEDLINE on STN DUPLICATE 67
ACCESSION NUMBER: 88317012 MEDLINE
DOCUMENT NUMBER: 88317012 PubMed ID: 3166071
TITLE: Cytogenetic analysis of melanocytes from premalignant nevi and melanomas.
AUTHOR: Cowan J M; Halaban R; Francke U
CORPORATE SOURCE: Department of Human Genetics, Yale University School of Medicine, New Haven, CT 06510.
CONTRACT NUMBER: CA-04679 (NCI)
CP-21037 (NCI)
GM-26105 (NIGMS)
SOURCE: JOURNAL OF THE NATIONAL CANCER INSTITUTE, (1988 Sep 21) 80 (14) 1159-64.
Journal code: 7503089. ISSN: 0027-8874.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198810
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 20000303
Entered Medline: 19881013
AB We karyotypically analyzed cultured melanocytes from a variety of lesions, including congenital and dysplastic nevi, primary melanoma, and metastatic melanoma. The cells derived from congenital nevi had normal karyotypes, as did 22 of the 26 cultures derived from dysplastic nevi. The karyotypes of melanocytes from primary and metastatic melanomas were all abnormal. The only chromosome change in common between the nevi with abnormal karyotypes and the melanomas was the loss of one copy of chromosome 9 (two of four nevi and four of 11 melanomas, including three from the same patient) or the loss of the short arm of chromosome 9, especially of region 9pter-p22 (three of 11 melanomas). We suggest that deletion of a gene or genes on 9p, possibly interferon genes, is an initial step in the malignant transformation of melanocytes.

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(FILE 'HOME' ENTERED AT 07:17:13 ON 29 OCT 2003)

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 07:17:56 ON 29 OCT 2003
L1 4335 S SPITZ OR BENIGN (3A) SKIN
L2 220 S L1 AND (GAIN OR LOSS)
L3 83 DUP REM L2 (137 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 07:23:28 ON 29 OCT 2003

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 07:25:46 ON 29 OCT 2003
L4 26902 S NEVI OR NEVUS
L5 915 S L4 AND (GAIN OR LOSS)
L6 261 S L5 AND CHROMOSOME
L7 137 DUP REM L6 (124 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 07:27:36 ON 29 OCT 2003

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 07:28:43 ON 29 OCT 2003

FILE 'STNGUIDE' ENTERED AT 07:28:43 ON 29 OCT 2003

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 07:30:07 ON 29 OCT 2003

FILE 'STNGUIDE' ENTERED AT 07:30:07 ON 29 OCT 2003

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L7 ANSWER 8 OF 12 MEDLINE on STN
ACCESSION NUMBER: 2000062984 MEDLINE
DOCUMENT NUMBER: 20062984 PubMed ID: 10594753
TITLE: Molecular cytogenetic analysis of **Spitz nevi** shows clear differences to **melanoma**.
AUTHOR: Bastian B C; Wesselmann U; Pinkel D; Leboit P E
CORPORATE SOURCE: Cancer Genetics Program, Cancer Center, University of California San Francisco, 94143-0808, USA..
bastian@cc.ucsf.edu
SOURCE: JOURNAL OF INVESTIGATIVE DERMATOLOGY, (1999 Dec) 113 (6) 1065-9.
Journal code: 0426720. ISSN: 0022-202X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200001
ENTRY DATE: Entered STN: 20000124
Last Updated on STN: 20000124
Entered Medline: 20000113

AB **Spitz nevus** is a **benign** neoplasm of melanocytes that can be difficult or impossible to distinguish from **melanoma** by clinical and histopathologic examination. We studied genomic DNA from 17 **Spitz nevi** by comparative genomic hybridization (CGH). Thirteen lesions showed no chromosomal aberrations, three cases had a **gain** involving the entire p-arm of chromosome 11, and one case showed a **gain** of chromosome 7q21-qter. Fluorescence in situ hybridization (FISH) on lesional tissue with a probe for the p-arm of chromosome 11 showed 6-10 p-arm signals per nucleus in those cases with a CGH-detected **gain** of chromosome 11p. One case with a normal CGH profile also showed increased copy number of 11p by FISH. Thus, the majority of **Spitz nevi** have a normal chromosomal complement at the level of CGH resolution; however some may contain **gains**, with 11p apparently being the most frequently involved location. These findings differ significantly from the previously reported changes in primary cutaneous **melanoma**, which show frequent deletions of chromosomes 9p (82%), 10q (63%), 6q (28%), and 8p (22%), as well as **gains** of chromosomes 7 (50%), 8 (34%), 6p (28%), 1q (25%) by CGH analysis. These clear differences in the location and frequencies of chromosomal aberrations in **Spitz nevi** and primary cutaneous **melanomas** could represent a basis for developing adjunctive techniques for refining accuracy in the difficult differential diagnosis of spitzoid **melanocytic** neoplasms.

L7 ANSWER 3 OF 12 MEDLINE on STN
ACCESSION NUMBER: 2002062077 MEDLINE
DOCUMENT NUMBER: 21645135 PubMed ID: 11787857
TITLE: Spitzoid malignant **melanoma** with lymph-node metastasis. Is a copy-number **loss** on chromosome 6q a marker of malignancy?
AUTHOR: Mihic-Probst D; Zhao J; Saremaslani P; Baer A; Komminoth P; Heitz P U
CORPORATE SOURCE: Department of Pathology, University Hospital, Zurich, Switzerland.. daniela@mihic@pty.usz.ch
SOURCE: VIRCHOWS ARCHIV, (2001 Dec) 439 (6) 823-6.
JOURNAL code: 9423843. ISSN: 0945-6317.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20020125
Last Updated on STN: 20020128
Entered Medline: 20020125

AB Distinction of spitzoid malignant **melanomas** (SMM) from **Spitz nevi** may be difficult or even impossible on the basis of conventional histology. In this report, a patient suffering from a primary lesion diagnosed as a **Spitz nevus** and a metastatic malignant **melanoma** approximately 4 years thereafter is described. A diagnosis of SMM was made subsequently upon review of the primary lesion. In the present analysis, we used comparative genomic hybridization (CGH) to define markers characteristic of SMM. The primary lesion revealed deletions on chromosomes 6q and 9p. In the metastasis, additional deletions on chromosomes 10p and 10q and **gains** of chromosome 7 were found. To our knowledge, no chromosomal aberration on chromosome 6 was hitherto demonstrated in **benign melanocytic nevi**. Findings reported in the literature suggest that human **melanoma** metastasis suppressor gene maps to 6q. In contrast, **losses** on chromosome 9p seem to be an early event in the development of **melanoma**. However, they are not only found in **melanomas** but are occasionally present in **Spitz nevi** as well as in **atypical nevi**. The CGH result with deletion of 6q in this difficult to diagnose primary **melanocytic** lesion strongly supports the diagnosis of malignant **melanoma**. To demonstrate the reliability of **loss** on chromosome 6q as a marker of SMM, a larger number of lesions must be investigated.